

Storage Temperatures Necessary to Maintain Cheese Safety

JAY RUSSELL BISHOP and MARIANNE SMUKOWSKI*
Wisconsin Center for Dairy Research, University of Wisconsin
1605 Linden Drive, Madison, WI 53706-1565, USA

SUMMARY

Available information on bacterial pathogen growth, stasis, and death in cheeses was reviewed and evaluated to determine storage temperatures necessary to maintain product safety. In view of the variety and large volume of cheeses consumed throughout the world, the incidence of foodborne outbreaks associated with cheeses is extremely low. Research revealed that the inherent characteristics of most cheeses create a hostile environment for bacterial pathogens, especially at elevated ripening and storage temperatures. Therefore, it is recommended that the following cheeses, manufactured in the United States with pasteurized or heat treated ($\geq 63^{\circ}\text{C}$ for ≥ 16 seconds) milk, should be exempt from refrigeration requirements during ripening, storage, shipping, and display: Asiago (medium and old), Cheddar, Colby, Feta, Monterey Jack, Muenster, Parmesan, Pasteurized process, Provolone, Romano, and Swiss/Emmentaler. It must be stressed that the manufacture of these cheeses must be done under the proper conditions of Good Hygiene Practices, Good Manufacturing Practices, and HACCP principles, and according to CFR requirements. In addition, the natural cheeses must include active cultures, and the storage and display temperatures must not exceed 30°C .

INTRODUCTION

Temperature-dependent storage of most foods has three major roles – to allow for curing/ripening of foods that contain added active starter cultures and enzymes, to prevent quality defects, and to control pathogen growth. In making decisions on whether a food requires time/temperature control for safety, the properties of the food itself must be considered (3). The role of temperature-dependent aging and storage is similar for cheese and for other foods, but the targets differ significantly because of unique inherent characteristics of the finished food product.

Transformation of chalky, acid-tasting curd into ductile, full-flavored cheese is accomplished during ripening through the action of milk enzymes, rennet, and various organisms in the cheese, including those in the starter culture. The biochemical changes that occur during cheese ripening are complex and involve fermentation of the carbohydrate; hydrolysis of fats and proteins with subsequent decarboxylation, deamination, and/or hydrogenation; and production of carbonyls, nitrogenous compounds, fatty acids, and sulfur compounds, all of which contribute to the overall body, texture, and flavor of the final product (63). These inherent characteristics also create a hostile environment for pathogens (25). This re-

A peer-reviewed article

*Author for correspondence: Phone: 608.265.6346; Fax: 608.262.1578
E-mail: msmuk@cdr.wisc.edu

view of scientific information on pathogen death and growth in cheeses at various storage temperatures will determine parameters necessary to ensure safety of cheeses in the marketplace. The United States cheese industry advocates the use of a science-based approach for assessing the risk posed by ready-to-eat foods for possible transmission of pathogens in the food supply (24). Applying HACCP principles enhances the manufacture of safe cheese (35).

In view of the variety and large volume of cheese consumed throughout the world, the incidence of outbreaks of food poisoning and foodborne disease associated with cheese are extremely low (36). Epidemiology studies of cheese-related outbreaks in the United States, Canada, and Europe have found no outbreaks linked to hard Italian varieties, e.g., Parmesan, Romano, and Provolone. Varieties such as Cheddar and Swiss were infrequently involved (38). In general, very few documented illness outbreaks have been linked to consumption of properly ripened hard cheese. Therefore, time/temperature control of hard cheese is primarily needed not for safety reasons, but to maintain the organoleptic quality of cheese (3).

INHERENT CHARACTERISTICS OF SAFE DAIRY FOODS

Numerous researchers have reported bactericidal and/or bacteriostatic effects on pathogenic bacteria in foods because of reduced moisture, low water activity, low pH as the result of organic acid production, salt, heat treatment, competing flora, biochemical metabolites, bacteriocins, and ripening, either singly or as part of hurdle technology (1, 3, 5, 6, 10, 11, 13, 15, 17, 22, 25, 26, 29, 34, 36, 37, 38, 39, 40, 43, 45, 48, 49, 51, 58, 59, 64, 65, 66, 68, 69, 70, 76). Refrigeration cannot be depended upon to reduce the number of pathogens, as it has been proven that *Listeria monocytogenes* (*L. monocytogenes*) and other psychrotrophic pathogens are capable of growth at these temperatures. Therefore, other factors, such as diligence with regard to good hygiene practices by the food industry, must be responsible for the lack of pathogen growth in fermented dairy foods. Results also confirm the low frequency of contamination by *L. monocytogenes* of pasteurized fluid milk products sold in the United States (24).

INHERENT CHARACTERISTICS OF CHEESE

Cheeses are one of the oldest types of prepared foods. Cheesemaking provided human kind with a means of concentrating and preserving milk at a time when refrigeration was unknown and principles of food preservation were vague empirical concepts at best (52).

The vast majority of cheese manufactured in the United States is made from pasteurized or heat-treated milk, which renders the product free of most pathogens (38, 39, 40). The inherent characteristics of cheeses made with starter culture addition provide multiple hurdles that inhibit pathogen growth (3, 47). A multiplicity of practices other than pasteurization or heat-treatment also contribute significantly to the microbiological safety of cheese (10, 11, 38). Some practices, such as milk quality management, lactic culture protocols, pH control, salt addition, and controlled curing conditions, are established technologies (38). Other factors may include natural inhibitory substances (e.g., lysozyme), starter metabolites and fermentation by-products (e.g., nisin), including organic acids (e.g., lactic, acetic, propionic, and formic). Water activity/moisture content imposes additional detrimental effects on foodborne pathogens during the manufacturing and ripening of cheese (10, 11, 38, 66).

During the manufacture of semi-soft, hard, and very hard cheeses, the cheese is subjected to relatively long exposure to ideal incubation temperatures for bacteria. For example, Cheddar and related varieties are maintained at 31–39°C during manufacture and are formed or hooped at temperatures in the 32–37°C range. Many Cheddar-type cheeses are cured or aged at temperatures up to 15.6°C. Swiss cheese is held for a period of 4–8 weeks at a temperature of 22.2–23.3°C to develop the characteristic eyes and flavor. If storage of Cheddar and Swiss cheese at room temperature had any inherent detrimental effect on safety of these cheeses, then neither would be safe to consume (51).

Specifically for *L. monocytogenes*, numerous studies suggest that the composition of cheese, ripening and storage conditions, lactic acid cultures, pH, salt, and moisture concentration influence its survival and growth (15, 29, 39, 40, 43). The fate of *L. monocytogenes* and other foodborne pathogens during cheese ripening is determined by the microbiological, biochemical, and physical properties

of the particular cheese (43, 64). Thus, cheese is a very complex system, with the following factors acting simultaneously to determine the behavior of *L. monocytogenes* during ripening: (a) type, amount, and activity of starter culture; (b) pH as determined by concentrations of lactic, acetic, formic, and other acids; (c) presence of hydrogen peroxide, diacetyl, and various antimicrobial agents (Nisin, diplococcin, and other bacteriocins); (d) levels of nutrients, salt, moisture, and oxygen; and (e) the cheese ripening temperature (64).

Fermentation is an age-old food preservation method used to inhibit the growth and survival of pathogenic bacteria (48). Lactic acid bacteria commonly used to produce fermented dairy products are antagonistic to foodborne pathogens and will either inhibit their growth or inactivate them (5, 13, 36, 59, 66, 70). In addition, research has shown that some starter cultures are detrimental to food spoilage organisms as well as various pathogens in these products (1, 17, 22, 51, 58, 69, 76). Responsible for this action are metabolites such as lactic and other acids, diacetyl, hydrogen peroxide, and various antibiotic-like substances produced by lactic acid bacteria, which are probably synergistic (34, 36, 37, 45, 49, 66).

Examples of pathogens that are susceptible to inactivation or growth inhibition by metabolites of lactic acid bacteria include *Salmonella* Typhimurium, enteropathogenic *Escherichia coli*, *Staphylococcus aureus*, and *L. monocytogenes* (66). Growth of *L. monocytogenes* is always inhibited appreciably in lactic acid cultured product when compared to that of the control, no matter how high the final pH of the fermented milk. Even when the final pH dropped only to 5.99, growth of the pathogen was inhibited by 84% relative to the control (65). This suggests that factors other than the hydrogen ion concentration are involved in the inhibition of *L. monocytogenes* by lactic acid bacteria (65). These observations have been documented by other researchers, who noted that lactic cultures inhibited pathogens such as salmonellae and staphylococci, even when pH was controlled at 6.6 (26). Modern lactic culture technology for cheese manufacturers has virtually eliminated *Staphylococcus*-caused outbreaks involving cheese (40). Vigorous starter growth should protect fermented milk products against the growth of pathogens and the formation of staphylococcal enterotoxin (36). Mathew and Ryser (48) reported increased injury of healthy *L. monocytogenes* cells during

TABLE I. Model *L. monocytogenes* exposure of cheese (2001)

	Contamination	Retail	Home storage	Home storage
Cheese	Frequency	Contamination	Growth rate	Time
Cheddar	Low	Low	Low	Long
Colby	Low	Low	Low	Long
Feta	Moderate	Moderate	Low	Long
Monterey Jack	Low	Low	Low	Long
Mozzarella	Low	Low	Moderate	Long
Parmesan	Low	Low	Low	Long
Processed	Low	Low	Moderate	Long
Provolone	Low	Low	Low	Long
Swiss	Low	Low	Low	Long

The evaluation revealed that there was a very low risk for listeriosis by Feta cheese, heat-treated natural and process cheeses, and aged cheeses (77).

Obtaining more information from research, industry, and regulatory experience, FDA/USDA (78) updated their *L. monocytogenes* risk analysis in 2003 with the following results (Table 2).

fermentation; at the end of the 24-h fermentation period, > 90% of the healthy *L. monocytogenes* cells were injured. Additionally, at the end of the product's shelf life, > 99% of the initial population was injured, and no significant decrease in the percentage of injury was observed. It was also discovered that the presence of *L. monocytogenes* did not adversely affect the growth of the starter culture at any inoculation level (48). Gengeorgis et al. (25) demonstrated that non-soft cheeses made with the use of starter cultures and pH values of ≤ 5.5 , as well as processed cheeses, will not support growth of *L. monocytogenes* at 4 to 30°C if the cheeses are contaminated from raw foods after the consumers open packages. Rapid acid production is the principal factor responsible for the elimination of pathogens from semi-hard cheese. The use of an effective starter culture is not only critical for preventing growth of pathogens, but also essential for the production of good quality cheese (6). The preservative effect of lactic acid bacteria can be attributed partly to the activation of the lactoperoxidase system and partly to bacteriocins (4).

Temperatures of curd cooking and aging/curing/ripening/storage have an impact on pathogen growth and survival in cheese. In hard cheese types with

higher curd cooking temperatures, growth is slight (68). There is considerable evidence showing that certain cheeses do not support growth of pathogens during the aging process and subsequent storage (11). A review of the literature related to the potential for growth of pathogens in hard cheeses that are aged for at least 60 days shows that such growth is not likely to occur because of factors inherent to these cheeses (31). Pathogens that survive the manufacturing process decrease faster at higher storage temperatures (14). The death rate of *Salmonella* in Samsøe cheese was slower at 10–12°C than at 16–20°C (36). It has been concluded that, for traditionally made hard cheeses, time/temperature control for safety is not required (3).

In most cheese varieties, salt concentrations attain levels of 1.6–3.0% of the total weight of the cheese, which would not affect most of the pathogenic bacteria in cheese. But it must be realized that salt is dissolved in the aqueous phase of the cheese only, the actual site of bacterial growth. Given the respective calculated values, salt concentrations in the aqueous phase reach levels of 2.2–6.5% or higher and will, in fact, at least slow down the growth rate of most bacteria and even have a detrimental effect on the more sensitive ones (68).

Where scientific data do not exist, all the inherent characteristics of cheese can serve as criteria in determining potential growth of pathogens by the use of mathematical modeling (16, 72, 79, 83). When two or more of these criteria are combined, the resultant effect is an additional hurdle to the outgrowth of pathogens of concern. It is this effect that makes it possible to store certain cheeses safely beyond either one of the two Food Code criteria for date marking and refrigeration (i.e., 7 days at 5°C or 4 days at 7.2°C). This led the US Food and Drug Administration to issue, on December 15, 1999 (11), a letter suggesting that regulatory agencies use their discretionary authority and defer enforcement action regarding date marking aged hard cheeses. In that letter, FDA granted a formal interpretation to the Food Code that hard and semisoft aged cheeses and pasteurized process cheese, each manufactured according to 21 CFR 133 as specifically cited above and maintained under refrigeration, are exempt from the Food Code's date marking provision related to refrigerated, ready-to-eat, potentially hazardous food. This interpretation has subsequently been incorporated into state statutes, such as Wisconsin's (2). Feta cheese was later added to this exemption list by FDA (in the case of Iowa Dept. Health vs. Shullsburg Creamery).

TABLE 2. Model *L. monocytogenes* exposure of cheese (2003)

	Contamination	Retail	Home storage	Home storage
Cheese	Frequency	Contamination	Growth rate	Time
Cheddar	Low	Low	Low	Long
Colby	Low	Low	Low	Long
Feta	Moderate	Moderate	Low	Long
Monterey Jack	Low	Low	Low	Long
Mozzarella	Low	Low	Moderate	Long
Muenster	Moderate	Low	Low	Long
Parmesan	Low	Low	Low	Long

The FDA/USDA evaluation classified cheeses as follows:

- Fresh soft – Queso fresco, Queso de Crema, Queso de Puna
- Soft unripened (> 50% moisture) – Cottage, cream, Ricotta
- Soft ripened (> 50% moisture) – Brie, Camembert, Feta, Mozzarella
- Semi-soft (>39–50% moisture) – Blue, Brick, Monterey Jack, Muenster, Provolone
- Hard (≤ 39% moisture) – Cheddar, Colby, Parmesan, Processed

SPECIFIC CHEESES AND THEIR INHERENT CHARACTERISTICS

Cheeses are typically categorized according to their moisture content:

- Soft ≥ 50%
- Semi-soft > 39 – < 50%
- Hard ≤ 39% (4, 22)

Hard and semi-soft cheeses are the focus of this research review.

Research by Gengeorgis and colleagues (25) has yielded results indicative of those obtained by other researchers, which prove death of pathogens in nonsoft cheeses stored at various temperatures. In this study, 49 market cheeses representing 24 varieties were purchased commercially. Cheeses were inoculated with 10⁴ cells of *L. monocytogenes* per square cm. The inoculum was a cocktail of 5 strains — Scott A, V7, RM-1, VPH1, VPH2. Inoculated cheeses were stored at 4, 8 and 30°C for up to 36 hours. Certain cheeses (Queso Fresco, Panela Ranchero, Ricotta, Teleme, Brie, Camembert, and Cottage) supported *Listeria* growth in cheese at one of the storage temperatures. Cheeses not supporting growth but caus-

ing gradual death at all temperatures included Cotija, cream, blue, Cheddar, Monterey Jack, Swiss, Colby, string, Provolone, Muenster, Feta, and Kasserri with pH values of 4.3–5.6; process cheese (pH 5.7–6.4); and Limburger cheese (pH 7.2). Overall, this study demonstrated that nonsoft cheeses made with the use of starter cultures and at pH values of ≤ 5.6, as well as processed cheeses, will not support growth of *L. monocytogenes* at 4–30°C if contaminated from raw foods (meat, poultry, fish, vegetables) after the opening of the packages by consumers. In all cheeses that caused gradual death (Cotija, cream, Blue, Cheddar, Monterey Jack, Swiss, Colby, Provolone, Muenster, Feta, Kasserri, Process, Limburger), death at 30°C was greater than or equal to death at 4°C.

Asiago (medium and old)

Medium and old Asiago (aged at least 6 months and 12 months, respectively) are hard cheeses with characteristics very similar to those of Parmesan. FDA has previously exempted these cheeses from date-marking (11) and stated that hard cheeses aged at least 60 days are not likely to support pathogen growth (31).

Bachman and Spahr (6) found that Swiss-type hard cheeses are hygienically safe and that the technology used in manufacturing these cheeses does not support growth of pathogens and leads to a more rapid rate of death.

Cheddar

Cheddar is a hard cheese that does not support *L. monocytogenes* growth and that causes gradual death at all temperatures (25). This finding is confirmed by an FDA correspondence (11) and also agrees with work by Ryser and Marth (61), who reported that growth of *L. monocytogenes* during Cheddar cheese manufacture appeared to be inhibited by proper acid development resulting from an active starter culture. Behavior of other pathogens during Cheddar manufacture and ripening show similar results. With normal starter activity, inoculated *Staphylococcus aureus* died rapidly (60), as did *Yersinia enterocolitica* (67). Norholt (54) illustrated die-off of *Salmonella* spp. after 2 weeks. Wood et al. (84) found that, of 11 vats of *Salmonella*-contaminated Cheddar cheese curd, only 2 remained positive in the finished cheese immediately after manufacture. In 1 and 4 months,

these 2 vats were clear of the inoculated *Salmonella*. This result is supported by studies of Goepfert et al. (28) and Hargrove et al. (32) in artificially inoculated Cheddar. Both groups found a 75–80% reduction in *Salmonella* after hooping and pressing during manufacture.

Numerous researchers have reported kill of pathogens at higher ripening and storage temperatures. *Salmonella* spp. survived longer when Cheddar cheese was stored at 4.5°C rather than 10°C (82). In general, a low pH and a high ripening temperature result in a higher inactivation rate for pathogenic organisms (61). Using stirred-curd Cheddar cheese, Goepfert et al. (28) showed that the number of *S. Typhimurium* decreased by a factor of 10,000 during 10–12 weeks of ripening at 13°C, whereas a similar decrease required 14–16 weeks at 7.5°C. Park et al. (58) reported that salmonellae survived during ripening of Cheddar cheese for up to 7 months at 13°C and 10 months at 7°C. Ryser and Marth (61) reported an inactivation rate of *L. monocytogenes* 0.9 logs less at 6°C than at 13°C. International Dairy Federation researchers demonstrated that the decrease in numbers of staphylococci in Cheddar was greater at higher temperatures (10°C and 13°C) than at 7°C (36).

Colby

Colby is a hard to semi-soft cheese that does not support *L. monocytogenes* growth and causes gradual death at all temperatures (25), a finding confirmed by an FDA correspondence (11). Various researchers studying the behavior of inoculated pathogens during Colby cheese manufacture and ripening determined that *E. coli* generally decreased over a period of weeks and was not detected after 4–6 weeks (41) and that numbers of *Y. enterocolitica* generally decreased over a period of weeks at 3°C (51). Yousef and Marth (85) found that, early in storage of Colby cheese, numbers of *Listeria* in the cheese remained relatively constant for a time that depended on the strain used. Numbers of *Listeria* in cheese decreased steadily thereafter at a rate that depended mainly on composition of the cheese. It should be noted that 2 of the 6 lots of cheese manufactured in this study had moisture levels higher than CFR specifications. IDF researchers demonstrated that the decrease in numbers of staphylococci in Colby was greater at the higher temperatures (10°C and 13°C) than at 7°C (36).

Feta

The Greek regulatory standard for Feta cheese stipulates that it cannot contain more than 56% moisture and less than 43% FDM. No standard exists for the amount of salt, but the salting procedure is described in this regulation. Commercial Feta produced in Greece normally contains about 2.5% salt (75). Currently, there is no US standard of identity for Feta, a soft ripened cheese that does not support *L. monocytogenes* growth and that causes gradual death at all temperatures (25, 55). Other experiments have shown that *Listeria* not only failed to grow in Feta but was gradually inactivated in whey and skim milk brine containing 12% salt (NaCl) (57). Papageorgiou and Marth (55) observed that the pH value of 2-day old Feta cheese decreased to 4.6, after which the growth of *L. monocytogenes* ceased.

Monterey Jack

Monterey Jack is a hard to semi-soft cheese which does not support *L. monocytogenes* growth and causes gradual death at all temperatures (25). Other than this referenced study, there exists little published research with this cheese. However, it is very similar, with regard to pH, aqueous NaCl, and moisture, to other cheeses that have been heavily studied and proven not to support pathogen growth.

Mozzarella

Mozzarella is a soft to semi-soft cheese that has a manufacturing protocol detrimental to bacteria. Buazzi et al. (9) found that the typical cooking of Mozzarella curd at 40°C for 30 min caused a 38% decrease of *L. monocytogenes*, compared to numbers of the pathogen in curd after cutting. Placing of curd in hot water (77°C) and stretching for 3–4 min caused complete demise of the pathogen. The curd temperature during stretching was 58–65°C. In conclusion, no *L. monocytogenes* was found in the cheese at the end of stretching, start of brining, and end of storage. The heat treatment given to the curd freed the product of *L. monocytogenes*, even though the curd initially contained approximately 6.2×10^4 cells of the pathogen per g. Ryser and Marth (64) reported that the heat treatment given to Mozzarella cheese curd is clearly sufficient to inactivate small numbers of *L. monocytogenes* that might be present. Villani et al. (81) found similar results during manufacture of traditional Mozzarella cheese from buffalo milk.

Stecchini et al. (71) addressed the issue of post-process contamination by inoculating the surface and packaging fluid of Mozzarella cheese with *L. monocytogenes* and then storing the product at 5°C for 21 days. Under these conditions, numbers of *L. monocytogenes* increased about 10,000-fold. Mozzarella was implicated in an outbreak of salmonellosis in 1984. Post-processing contamination was thought to have caused the outbreak (19).

Muenster

Muenster is a semi-soft cheese that does not support *L. monocytogenes* growth and causes a gradual death at all temperatures (25). Other than this referenced study, there exists little published research with this cheese. However, it is very similar in pH, aqueous NaCl, and moisture, to other cheeses that have been heavily studied and proven not to support pathogen growth.

Parmesan

Parmesan is a hard cheese ripened at 12.8°C for 10 months, which does not support *L. monocytogenes* growth and which causes gradual death at all temperatures. No outbreaks in the United States have implicated any Italian-type hard cheeses, including Parmesan. This unblemished safety record may reflect conditions during manufacture and curing that inhibit or destroy pathogens (40). Yousef and Marth (86) observed that, during Parmesan cheese ripening, numbers of *L. monocytogenes* decreased almost linearly and faster than reported for other hard cheeses. *L. monocytogenes* was not detected in cheese after 2–16 weeks of ripening, depending on the strain of the pathogen and the lot of cheese. Parmesan made in this study was not a favorable medium for survival of *L. monocytogenes*. Decreased viability of the pathogen in Parmesan is probably related to a combination of factors, including (a) action of lipase added to the milk; (b) heat treatment that the curd receives during cheesemaking; and (c) lower moisture content and water activity of the fully ripened cheese.

Parmesan is more acidic than other cheeses, with a much lower water activity that inhibits microbial growth (35, 44). Pathogenic bacteria vary just as widely as the cheeses they contaminate, and their survival characteristics are equally varied. For example, Brie stored under refrigeration will support the growth of *L. monocytogenes*, while Parmesan stored at near-ambient temperature will not (35).

TABLE 3. Summary of data on cheeses reviewed, and compositional calculations (21, 68, 75)

Cheese Type	Typical % H ₂ O	CFR Limit % H ₂ O	A _w	Typical pH	Typical % NaCl	Typical % Aqueous NaCl	% FDM **	Active Culture	Age at sale (days)	Other inherent characteristics	Pathogen Kill+
Asiago	32-34	35	0.93	5.2-5.5	1.9-2.2	5.75	45	Thermophile	180-365	A/S Temp*	Ah, Cj, Ec, Lm, P, Sa, Sta, Ye
Cheddar	38	39	0.95	5.2	1.7	4.47	52	Mesophile	15-1,000	A/S Temp*	Lm, Sa, Sta, Ye
Colby	39	40	0.95	5.2	1.7	4.36	52	Mesophile	15-80	A/S Temp*	Ec, Lm, Sta, Ye
Feta	53	NA	0.95	4.5	3.0	5.66	29-52	Mesophile	7-90	A/S Temp*	Lm
Monterey Jack	38-42	44		5.25	1.7	4.05-4.47	52	Mesophile	15-150	A/S Temp*	Lm
Mozzarella	45-52	45-52		4.9-5.4	1.6	3.07-3.56	52	Thermophile	5-150	Hot water/steam treatment	Lm kill cook/stretch Lm, Sa growth
Muenster	43	46	0.98	5.2	1.8	4.18	52	Thermophile	10-150	A/S Temp*	Lm
Parmesan	31	32	0.92	5.4	2.6	8.38	38	Thermophile	300-600	A/S Temp* Aged ≥300d High temp curd cook Lipase activity	Lm
Process (sliceable)	40		0.92	5.6	2.2	5.50	50	None	14-180	A/S Temp* Heated ≥150°F/≥30 sec	Clb, Ec, Lm, Sa, Sta
Provolone	42.5	45	0.91	5.2	1.8	4.24	45	Thermophile	15-150	A/S Temp*	Lm
Romano	33.5	34	0.92	5.3	2.2	6.57	40	Thermophile	150-180	A/S Temp*	Lm
Swiss / Emmentaler	38	41	0.97	5.6	1.2	3.16	43	Thermophile	61-300	A/S Temp*	Ah, Cj, Ec, Lm, Pa, Sa, Sta, Ye
Brick	43	44		5.3	1.6	3.72	52	Mesophile	7-50	A/S Temp*	Ec, Lm
Blue	43	46	0.97	6.0	2.5	5.82	52	Mesophile	61-240		Lm

* A/S Temp => Increased pathogen kill at elevated aging/storage temperatures.

** %FDM=> Percent fat in dry matter.

+ Ah – *Aeromonas hydrophils*, Cj – *Campylobacter jejuni*, Clb – *Clostridium botulinum*, Ec – *Escherichia coli* O157:H7, Lm – *L. monocytogenes*,

P – *Pseudomonas aeruginosa*, Sa – *Salmonella* sp., Sta – *Staphylococcus aureus*, Ye – *Yersinia enterocolitica*.

Pasteurized Process (21CFR133.169)

Pasteurized process cheese is a soft to semi-soft cheese that does not support *L. monocytogenes* growth and that causes gradual death at all temperatures (25, 27). Pasteurized processed cheese and related products have an excellent safety record in the United States (39). During the past 50 years, very few disease outbreaks have been attributed to contaminated pasteurized process cheese products (27). The combined effects of pH, moisture, and salt in standardized process cheese may inhibit vegetative pathogen growth in a way

similar to the mechanism of inhibition for *Clostridium botulinum* (73, 74). If a pasteurized processed cheese is intended for use at ambient temperature, pH, water activity (a_w), moisture content, and antimicrobials should be appropriately adjusted to inhibit botulinal toxin formation (3). During manufacture, the product is heated for ≥ 30 s at a temperature of ≥ 65.6°C; this is sufficient to eliminate vegetative organisms but not the spores of *Clostridium botulinum*. As a formulated safe product with regard to *C. botulinum*, the combinations of moisture, salt, and pH act as multiple hurdles to inhibit botulinal growth and toxin production (42, 73).

While studying pathogen survival in pasteurized process cheese slices, Glass et al. (27) reported that populations of *Salmonella* serotypes and *E. coli* O157:H7 decreased by an average of 1.3 and 2.1 log CFU/g, respectively, by 36 h. *Salmonella* serotypes decreased an additional 0.6 log CFU/g during the remaining 60 h. Populations of *L. monocytogenes* also decreased, although to a lesser extent, exhibiting approximately 0.6 log CFU/g reduction in 96 h. *S. aureus* levels remained relatively constant during the testing period and were below levels that support detectable enterotoxin production. At 30°C, the pasteurized process cheese slices

allowed survival but did not support growth of *S. aureus*, whereas populations of *L. monocytogenes*, *E. coli* O157:H7, and *Salmonella* serotypes decreased during the 96 h storage. Water activity appears to contribute significantly to the inhibition of pathogen growth in these cheese slices. The a_w of the tested formulations (0.92–0.93) was at or below the minimum required for growth of most foodborne pathogens. Although low a_w may inhibit pathogen growth in these formulations, the synergistic effect of moisture, salts, and pH, or another factor such as sorbate, may also contribute to the safety of the product. The results suggest that properly formulated pasteurized process cheese could be exempt from the potentially hazardous food category because it does not support the rapid and progressive growth of pathogens tested. The results of the study suggested that unopened packages of properly formulated pasteurized process cheese can be safely stored unrefrigerated for certain time periods (53). In fact, reducing storage temperatures has been reported to actually enhance survival of *E. coli* O157:H7 in acidified media, apple cider, and mayonnaise (33, 50, 87).

Provolone

Provolone is a semi-soft cheese that does not support *L. monocytogenes* growth and that causes gradual death at all temperatures (25). No outbreaks that implicated any Italian-type cheese, including Provolone (40), have been found in the United States. Other than this referenced study, little published research with this cheese exists. However, with regard to pH, aqueous NaCl, and moisture, it is very similar to other cheeses that have been heavily studied and proven not to support pathogen growth.

Romano

Romano is a hard cheese that does not appear to support *L. monocytogenes* growth. In the United States, no outbreaks have been found that implicated any Italian-type cheeses, including Romano (40). Other than this referenced study, there exists little published research with this cheese. However, it is very similar to other cheeses with regard to pH, aqueous NaCl, and moisture, which have been heavily studied and proven not to support pathogen growth.

Swiss / Emmentaler

Swiss/Emmentaler is a hard to semi-soft cheese that does not support *L. monocytogenes* growth and that causes

gradual death at all temperatures (25). This finding is confirmed by an FDA correspondence (11). The ripening temperature of Swiss cheese is comparatively high (22°C). Buazzi et al. (10) reported a sharp decrease in numbers of *L. monocytogenes* during brining of Swiss blocks (7°C for 30 h). The population of *L. monocytogenes* continues to decrease during cheese ripening. *Listeria* was not detected after 80, 77, and 66 days of ripening of Swiss cheese made from inoculated milk. Bachmann and Spahr (6) discovered none of the inoculated potentially pathogenic bacteria, except for low numbers of *S. aureus*, could be found in the experimental Swiss cheese 1 day after manufacturing. All subsequent determinations showed that the cheese was free from potentially pathogenic bacteria and toxins. Baumgartner et al. (8) previously reported the same behavior of *S. aureus* in Emmentaler cheese. Bachmann and Spahr (6) also found that even in poor quality cheese that had been inoculated with *E. coli* and was exhibiting early blowing, no *E. coli* could be detected at the end of ripening. Additionally, results showed that 1 week after manufacturing, the inoculated pathogens (*Aeromonas hydrophila*, *Campylobacter jejuni*, *E. coli*, *L. monocytogenes*, *Pseudomonas aeruginosa*, *Salmonella* spp., *S. aureus*, and *Y. enterocolitica*) could no longer be detected.

El-Shenawy and Marth (18) suggested that production of propionate by eye-forming bacteria may have contributed to the demise of *L. monocytogenes* in Swiss cheese. In other work, < 2,000 ppm of sodium propionate inhibited growth of *L. monocytogenes* at pH 5.0 (10). At pH 5.0 and 3,000 ppm sodium propionate, the *Listeria* population decreased 1,000-fold during 67 days of incubation at 35°C and disappeared after 78 days. A 60-day-old Swiss cheese typically contains 3,750 ppm propionic acid (46). Acetate may also play a major role in inactivating *L. monocytogenes* in Swiss cheese (10); more lactate is fermented to acetate and CO₂ than to propionate (12). The rapid decrease of the redox potential of Swiss cheese probably supports the inhibitory effect on pathogenic bacteria (54).

Generally, manufacturing technology of Swiss cheese does not support the growth of pathogenic bacteria (6, 10). Because of the synergistic effect of active antimicrobial enzyme systems in fresh raw milk, antagonistic starter culture flora, fast acidification, antimicrobial effect of lactic acid, and high curd cooking temperatures,

potentially pathogenic bacteria do not survive the manufacturing of Swiss cheese produced under good manufacturing practices. In addition, intense brining and ripening at elevated temperatures for at least 2 months eliminate the occurrence of the tested strains. Pathogens that may survive the manufacturing process decrease faster at higher storage temperatures (14). Swiss cheese appears to pose a very low risk for transmission of foodborne diseases (40).

Brick

Brick is a semi-soft cheese. In studies of the behavior of pathogens during Brick cheese manufacture and ripening, *L. monocytogenes* numbers decreased during 20–22 weeks of curing at 10°C (67), and *E. coli* grew during manufacture and then died off during curing (23).

Blue

Blue is considered a semi-soft cheese that has been proven to not support *L. monocytogenes* growth and that causes gradual death at all temperatures (25). Papageorgiou and Marth (59) reported that growth of *L. monocytogenes* ceased when the pH of blue cheese dropped below 5.0. Populations of *L. monocytogenes* decreased significantly ($P \leq 0.005$) during the first 50 days of ripening, by an average of 2.6 logs CFU/g compared to populations of 1-day old cheese. The high salt content in blue cheese is likely the main reason for the lack of growth of *Listeria*. Productions of fatty acids and methyl ketones derived from fatty acids via the beta-oxidation pathway, and their corresponding secondary alcohols, may contribute to the unfavorable environment for *L. monocytogenes* (32). Blue cheese on the market has a pH >5.0; therefore, conclusive pathogen death is not verified.

Soft / Hispanic

This category includes Queso Blanco, Queso Fresco, Ricotta, Teleme, Brie, Camembert, Panela, Ranchero, cream, and cottage. Gengeorgis et al. (25) evaluated the fate of *Listeria* as a post-processing contaminant and found that *Listeria* growth was primarily confined to high-moisture varieties, including Brie, Camembert, Ricotta, and the soft Hispanic cheeses, all of which had a pH ≥ 6.0 and low to moderate levels of salt in the moisture phase. Back et al. (7) noted that *L. monocytogenes* survived, and under most conditions multiplied, when inoculated directly into the cheese milk of laboratory-made Camembert cheese.

REGULATORY EVALUATION

In a series of correspondences, in a letter form as an inclusion to the US FDA Program Information Manual on retail Food Safety and in a subsequent correspondence (11, 31), FDA exempted the following cheeses from the date marking mandate within the US Food Code:

Asiago	Limburger
Blue	Monterey Jack
Brick	Muenster
Cheddar	Parmesan
Colby ($\leq 40\%$ moisture)	Pasteurized process
Edam	Provolone
Feta	Reggiano
Gorgonzola	Romano
Gouda	Sapsago
Gruyere	Swiss/Emmentaler

In 2001, FDA/USDA (77) conducted a risk analysis of foodborne outbreaks of *L. monocytogenes* from ready-to-eat foods (Table 1).

The evaluation revealed that there was a very low risk for listeriosis by Feta cheese, heat-treated natural and process cheeses, and aged cheeses (77).

Obtaining more information from research, industry, and regulatory experience, FDA/USDA (78) updated their *L. monocytogenes* risk analysis in 2003 with the following results (Table 2).

Utilizing a cluster analysis of predicted risk that takes into account the relative risk of listeriosis for the total population on a per serving and per annum basis, the following risk categories were developed for cheese:

- High risk – soft unripened cheeses (cottage, cream)
- Moderate risk – fresh soft cheeses (Queso Fresco) soft ripened cheeses (Brie, Camembert, Feta, Mozzarella) semi-soft cheese (Blue, Brick, Monterey Jack)
- Very low risk – hard cheeses (Cheddar, Swiss, Parmesan)
- Process cheeses

FDA/USDA actually decreased the predicted risk of soft ripened and certain semi-soft cheeses to “Moderate” due to increased use of pasteurized or otherwise heat-treated milk, and effective food safety control programs.

The very low risk cheeses have similar characteristics of being subjected to bactericidal treatment, having very low contamination rates, and possessing an inherent characteristic (or two) that either inactivates *L. monocytogenes* (hard

cheese) or prevents its growth (process cheese). As can be noted from this review, many more cheeses fit this category than recognized by USDA. The relative risk indices used may not give a clear picture of the range of risk potential that exists. The differential between per-serving risks associated with deli meats (relative risk rank of 1) and hard cheeses (relative risk rank of 23) is almost 10,000,000-fold (78).

CONCLUSIONS

Science-based data presented herein adequately illustrate the fact that most cheeses containing $< 50\%$ moisture (or more, in the case of Feta) and active lactic acid starter cultures, along with traditional levels of salt, pH, fat, etc., do not allow the growth of pathogens at temperatures between 4 and 30°C. In fact, in the vast majority of the cheeses, a higher temperature during ripening/aging and storage leads to significant bactericidal activity. A summary of the reviewed science and data is available in Table 3.

Mathematical models were generated using the USDA Pathogen Modeling Program, but given that this system is in nutrient broth, not in a limited moisture solid food (cheese), growth/death curves generated were meaningless. No other models reviewed were found to be appropriate.

RECOMMENDATIONS

For cheeses manufactured in the United States with pasteurized or heat-treated ($\geq 63^\circ\text{C}$ for ≥ 16 s) milk, under hygienic conditions outlined in Good Hygienic Practices, Good Manufacturing Practices, and HACCP systems, using active lactic acid cultures, and according to CFR specifications, the following cheese should be considered by regulatory agencies (FDA, USDA, state, local, etc.) exempt from any and all refrigeration requirements for aging, storage, shipping, and retail display, with a maximum temperature of 30°C:

Asiago (medium and old)
Cheddar
Colby
Feta
Monterey Jack
Muenster
Parmesan
Pasteurized process cheese
Provolone
Romano
Swiss / Emmentaler

If this exemption would apply only to pre-packaged cheeses, Parmesan and Romano, and possibly medium and old Asiago — because of their inherent characteristics — would not have to be pre-packaged for this refrigeration exemption. Soft/fresh Asiago, Blue, Brick, cream and Mozzarella require further investigation before a recommendation for exemption could be made.

There is one common thread among all the ripened cheeses evaluated (this would exclude Mozzarella); the curing/ripening/aging step is detrimental to bacterial pathogens, especially at elevated temperatures up to 30°C. Therefore, for safety purposes, refrigerated storage of the cheeses would appear to be unnecessary and possibly counterproductive.

ACKNOWLEDGMENTS

We are grateful to Dr. Kathy Glass of the Food Research Institute for providing valuable input into this paper. We are grateful to Charlie Guan, Kristen Houck and Karen Paulus of the Wisconsin Center for Dairy Research for their assistance.

REFERENCES

1. Ahmed, A. H., M. Moustafa, and T. A. El-Bassiony. 1986. Growth and survival of *Yersinia enterocolitica* in yogurt. *J. Food Prot.* 49:983–985.
2. Anonymous. 2001. Date marking of potentially hazardous, ready-to-eat foods. Wisconsin Foods Code Fact Sheet # 16, Wisconsin Dept Ag, Trade, Consumer Prot.
3. Anonymous. 2003. Analysis of microbial hazards related to time/temperature control of foods for safety-IFT/FDA Report on Task Order 4. Comprehensive Reviews in Food Science and Food Safety, Volume 2.
4. Asperger, H. 1986. Wirkungen von milchsäurebakterien auf andere mikroorganismen: Ein überblick. *Oesterr. Milchwirtschaft* 41, Beilage 1:1.
5. Babel, F. J. 1977. Antibiosis by lactic culture bacteria. *J. Dairy Sci.* 60:815–821.
6. Bachmann, H. P., and U. Spahr. 1995. The fate of potentially pathogenic bacteria in Swiss, hard and semi-hard cheeses made from raw milk. *J. Dairy Sci.* 78(3):476–483.
7. Back, J. P., S. A. Langford, and R. G. Kroll. 1993. Growth of *L. monocytogenes* in Camembert and other soft cheeses at refrigeration temperatures. *J. Dairy Res.* 60:421–429.

8. Baumgartner, H., P. Kaestli, and P. Weber. 1968. Das Verhalten von *Staphylococcus aureus* in Milch und bei der Herstellung von Emmentalerkase. *Schweiz Milchzeitung*. 94:993.
9. Buazzi, M. H., M. E. Johnson, and E. H. Marth. 1992. Fate of *L. monocytogenes* during the manufacture of Mozzarella cheese. *J. Food Prot.* 55(2): 80–83.
10. Buazzi, M. M., M. E. Johnson, and E. H. Marth. 1992. Survival of *L. monocytogenes* during the manufacture and ripening of Swiss cheese. *J. Dairy Sci.* 75(2):380–386.
11. Buchanan, R. L., E. L. Harden, and R. D. Beaulieu. 1999. Date marking of cheese. FDA Program Information Manual-Retail Food Safety.
12. Crow, V. 1986. Metabolism of aspartate by *Propionibacterium freudenreichii* subsp. *shermanii*: effect of lactate fermentation. *Appl. Environ. Microbiol.* 52:359–365.
13. Daly, C., W. E. Sandine, and P. E. Elliker. 1972. Interaction of food starter cultures and food-borne pathogens: *Streptococcus diacetylactis* versus food pathogens. *J. Milk Food Technol.* 35(6):349–357.
14. Daoism, J. Y., D. W. Warburton, and A. M. Sewell. 1985. *Salmonella* Typhimurium phage-type 10 from Cheddar cheese implicated in a major Canadian foodborne outbreak. *J. Food Prot.* 48(12):1062–1066.
15. Dominguez, L., J. F. F. Garayzabal, J. A. Vazquez, J. L. Blanco, and G. Suarez. 1987. Fate of *L. monocytogenes* during manufacture and ripening of semi-hard cheese. *Letts. Appl. Microbiol.* 34:95–100.
16. Duh, Y-H, and D. W. Schaffner. 1993. Modeling the effect of temperature on the growth rate and lag time of *L. innocua* and *L. monocytogenes*. *J. Food Prot.* 56(3):205–210.
17. Ehlers, J. G., M. Chapparo-Serrano, R. Richter, and C. Vanderzant. 1982. Survival of *Campylobacter fetus* subsp. *jejuni* in Cheddar and cottage cheese. *J. Food Prot.* 45:1018–1021.
18. El-Shenawy, M. A., and E. H. Marth. 1989. Behavior of *L. monocytogenes* in the presence of sodium propionate. *Intern. J. Food Microbiol.* 8(1):85–94.
19. Felip, G., and L. Toti. 1984. Food poisoning caused by Gram-negative bacteria in milk products. *Microbiol. Aliment. Nutr.* 2:251–256.
20. Food and Drug Administration. 2005. Cheese and cheese products. Definitions and standards under the Federal Food, Drug and Cosmetic Act. 21 Code of Federal Regulations 133.
21. Fox, P. F., T. P. Guinee, T. M. Cogan, and P. L. H. McSweeney. 2000. *Fundamentals of Cheese Science*. Aspen Publishers.
22. Frank, J. F., and E. H. Marth. 1977. Inhibition of enteropathogenic *Escherichia coli* by homofermentative lactic acid bacteria in skim milk. *J. Food Prot.* 40:749–753.
23. Frank, J. F., E. H. Marth, and N. F. Olson. 1978. Behavior of enteropathogenic *Escherichia coli* during manufacture and ripening of Brick cheese. *J. Food Prot.* 41:111–115.
24. Frye, C., and C. W. Donnelly. 2005. Comprehensive survey of pasteurized fluid milk produced in the United States reveals a low prevalence of *L. monocytogenes*. *J. Food Prot.* 68(5):973–979.
25. Gengeorgis, C., M. Carniciu, D. Dutulescu, and T. B. Farver. 1991. Growth and survival of *L. monocytogenes* in market cheeses stored at 4 to 30°C. *J. Food Prot.* 54(9):662–668.
26. Gilliland, S. E., and M. L. Speck. 1972. Interactions of food starter cultures and foodborne pathogens: Lactic streptococci versus staphylococci and salmonellae. *J. Milk Food Technol.* 35(5):307–310.
27. Glass, K. A., K. M. Kaufman, and E. A. Johnson. 1998. Survival of bacterial pathogens in pasteurized process cheese slices stored at 30°C. *J. Food Prot.* 61(3):290–294.
28. Goepfert, J. M., N. F. Olson, and E. H. Marth. 1968. Behavior of *Salmonella* Typhimurium during manufacture and curing of Cheddar cheese. *Appl. Microbiol.* 16(6):862–866.
29. Griffiths, M. W. 1989. *L. monocytogenes*: Its importance in the dairy industry. *J. Sci. Food Agric.* 47:133–158.
30. Gripon, J. C. 1987. Mould-ripened cheeses, pp. 121–149. In P. F. Fox (ed.), *Cheese: chemistry, physics and microbiology*, Vol. 2. Major Cheese Groups. Elsevier Appl. Sci., London.
31. Harden, B. 1999. FDA correspondence, April 5. Subject: Aged Hard Cheeses.
32. Hargrove, R. E., F. E. McDonough, and W. A. Mattingly. 1969. Factors affecting survival of *Salmonella* in Cheddar and Colby cheese. *J. Milk Food Technol.* 32:480–484.
33. Hathcox, A. K., L. R. Beuchat, and M. P. Doyle. 1995. Death of enterohemorrhagic *E. coli* O157:H7 in real mayonnaise and reduced-calorie mayonnaise dressing as influenced by initial population and storage temperature. *Appl. Environ. Microbiol.* 61:4172–4177.
34. Hawley, H. B. 1957. Nisin in food technology. I. *Food Manuf.* 32:370–376.
35. Institute of Food Science and Technology. 2005. Food safety and cheese. IFST: Current Hot Topics, Feb. 2.
36. International Dairy Federation. 1980. Behavior of pathogens in cheese. *IDF Bulletin* 122.
37. Jay, J. M. 1982. Antimicrobial properties of diacetyl. *Appl. Environ. Microbiol.* 44:525–532.
38. Johnson, E. A., J. H. Nelson, and M. E. Johnson. 1990. Microbiological safety of cheese made from heat-treated milk, Part I. Executive summary, introduction and history. *J. Food Prot.* 53(5):441–452.
39. Johnson, E. A., J. H. Nelson, and M. E. Johnson. 1990. Microbiological safety of cheese made from heat-treated milk, Part II. Microbiology. *J. Food Prot.* 53(6):519–540.
40. Johnson, E. A., J. H. Nelson, and M. E. Johnson. 1990. Microbiological safety of cheese made from heat-treated milk, Part III. Technology, discussion, recommendations, bibliography. *J. Food Prot.* 53(7): 610–623.
41. Kornacki, J. L., and E. H. Marth. 1982. Fate of nonpathogenic and enteropathogenic *Escherichia coli* during the manufacture of Colby-like cheese. *J. Food Prot.* 45(4):310–316.
42. Kosikowski, F. V., and V. V. Mistry. 1997. *Cheese and Fermented Milk Products*. 3rd ed., vol. I, p. 328–352. FV Kosikowski, Westport, CT.
43. Kovincic, I., I. F. Vujcic, M. Svabic-Vlahovic, M. Vulic, M. Gagoc, and I. V. Wesley. 1991. Survival of *L. monocytogenes* during the manufacture and ripening of Trappist cheese. *J. Food Prot.* 54(6):418–420.

44. Kozak, J., T. Balmer, R. Byrne, and K. Fisher. 1996. Prevalence of *L. monocytogenes* in foods: Incidence in dairy products. *Food Control* 7(4/5):215–221.
45. Kozak, W., J. Bardowski, and W. T. Dobrzanski. 1977. Lactostreptins-acid bacteriocins produced by *Lactic streptococci*. *J. Dairy Res.* 45:247–254.
46. Langsrud, T., and G. W. Reinbold. 1973/1974. Flavor development and microbiology of Swiss cheese – A review. *J. Milk Food Technol.* 36(12): 593–609.
47. Leistner, X. 1978. Hurdle effect and energy saving. W. K. Downey (ed.) *Food Quality and Nutrition*. Applied Science Publishers. London.
48. Mathew, F. P., and E. T. Ryser. 2002. Competition of thermally injured *L. monocytogenes* with a mesophilic lactic acid starter culture in milk for various heat treatments. *J. Food Prot.* 65(4):643–650.
49. Mathot, A. G., E. Beliard, and D. Thuault. 2003. *Streptococcus thermophilus* 580 produces a bacteriocin potentially suitable for inhibition of *Clostridium tyrobutyricum* in hard cheeses. *J. Dairy Sci.* 86(10): 3068–3074.
50. Miller, L. G., and C. W. Kasper. 1994. *Escherichia coli* O157:H7 acid tolerance and survival in apple cider. *J. Food Prot.* 57:460–464, erratum 57:645.
51. Moustafa, M. K., A. A. -H Ahmed, and E. H. Marth. 1983. Behavior of virulent *Yersinia enterocolitica* during manufacture and storage of Colby-like cheese. *J. Food Prot.* 46(4):318–320.
52. Nelson, J. H. 1983. Should cheese be refrigerated? *Dairy Food Sanit.* 3(10):372–375.
53. Nelson, J. H. 1984. Some regulatory compliance concerns of the cheese industry. *J. Dairy Sci.* 67:2108–2119.
54. Norholt, M. D., 1984. Growth and inactivation of pathogenic microorganisms during manufacture and storage of fermented dairy products: A review. *Neth. Milk Dairy J.* 38:135–150.
55. Papageorgiou, D. K., and E. H. Marth. 1989. Fate of *L. monocytogenes* during the manufacture, ripening and storage of Feta cheese. *J. Food Prot.* 52(2):82–87.
56. Papageorgiou, D. K., and E. H. Marth. 1989. Fate of *L. monocytogenes* during the manufacture and ripening of Blue cheese. *J. Food Prot.* 52(7): 459–465.
57. Papageorgiou, D. K., and E. H. Marth. 1989. Behavior of *L. monocytogenes* at 4 and 22°C in whey and skim milk containing 6 or 12% NaCl. *J. Food Prot.* 52(9):625–630.
58. Park, H. S., E. H. Marth, J. M. Goepfert, and N. F. Olson. 1970. The fate of *Salmonella* Typhimurium in the manufacture and ripening of low-acid Cheddar cheese. *J. Milk Food Technol.* 33:280–284.
59. Reiter, B. 1985. Interaction between immunoglobulins and innate factors such as lysozyme, lactoferrin, lactoperoxidase. J. Schaub (ed.) *compos. Physiol. Prop. Human Milk*, Proc. Intern. Workshop, Elsevier, Amsterdam pp. 271–284.
60. Reiter, B., B. G. Fewins, T. F. Fryer, and M. E. Sharpe. 1964. Factors affecting the multiplication and survival of coagulase positive *Staphylococci* in Cheddar cheese. *J. Dairy Res.* 31:261–272.
61. Ryser, E. T., and E. H. Marth. 1987. Behavior of *L. monocytogenes* during the manufacture and ripening of Cheddar cheese. *J. Food Prot* 50(1):7–13.
62. Ryser, E. T., and E. H. Marth. 1989. Behavior of *L. monocytogenes* during the manufacture and ripening of Brick cheese. *J. Dairy Sci.* 72(4):838–853.
63. Ryser, E. T., and E. H. Marth. 1991. Incidence and behavior of *L. monocytogenes* in cheese and other fermented dairy products. *L. monocytogenes*, Listeriosis and Food Safety. *L. monocytogenes* in Fermented Dairy Products, pp. 331–404.
64. Ryser, E. T., and E. H. Marth. 1999. Incidence and behavior of *L. monocytogenes* in cheese and other fermented dairy products. *L. monocytogenes*, Listeriosis and food safety, 2nd ed. Revised and expanded. *L. monocytogenes* in Fermented Dairy Products, pp. 411–503.
65. Schaak, M. M., and E. H. Marth. 1988. Behavior of *L. monocytogenes* in skim milk during fermentation with mesophilic lactic starter cultures. *J. Food Prot.* 51(8):600–606.
66. Schaak, M. M., and E. H. Marth. 1988. Interaction between lactic acid bacteria and some foodborne pathogens: A review. *Cultured Prod. J.* Nov: 14–20.
67. Schiemann, D. 1978. Association of *Yersinia enterocolitica* with the manufacture of cheese and occurrence in pasteurized milk. *Appl. Environ. Microbiol.* 36:274–277.
68. Spahr, U., and B. Url. 1994. Behaviour of pathogenic bacteria in cheese – A synopsis of experimental data. *IDF bulletin* 298:2–16.
69. Speck, M. L. 1971. Control of food-borne pathogens by starter cultures. *J. Dairy Sci.* 55:1019–1022.
70. Stadhouders, J., M. M. Cordes, and A. W. J. van Schouwenberg-van Foeken. 1978. The effect of manufacturing conditions on the development of *Staphylococci* in cheese: their inhibition by starter bacteria. *Neth. Milk Dairy J.* 32:193–203.
71. Stecchini, M. L., V. Aguilí, and I. Sarais. 1995. Behavior of *L. monocytogenes* in Mozzarella cheese in the presence of *Lactococcus lactis*. *Intern. J. Food Microbiol.* 25(3):301–310.
72. Sutherland, J. P., A. J. Bayliss, and T. A. Roberts. 1994. Predictive modeling of growth of *Staphylococcus aureus*: the effects of temperature, pH and sodium chloride. *Intern. J. Food Microbiol.* 21:217–236.
73. Tanaka, N., E. Traisman, P. Plantinga, L. Finn, W. Flom, L. Meske, and J. Guggisberg. 1986. Evaluation of factors involved in antibotulinal properties of pasteurized process cheese spreads. *J. Food Prot.* 49:526–531.
74. ter Steeg, P. F., and H. G. M. Cuppers. 1995. Growth of proteolytic *Clostridium botulinum* in process cheese products: II. Predictive modeling. *J. Food Prot.* 58:1108–1110.
75. The Food and Beverage Code 1971. Article 83. Modification, 1987. Newspaper of the Government of the Republic of Greece, Athens, July 3, 1987.
76. Tiwari, N. P., and I. P. Singh. 1964. Survival of pathogens in Dahi. I: Contamination of milk before the preparation of Dahi. *Indian J. Dairy Sci.* 17:97–101.
77. U.S. Department of Agriculture. 2001. Interpretive Summary: Draft assessment of the relative risk to public health from foodborne *L. monocytogenes* among selected categories of ready-to-eat foods. CFSAN/FDA, FSIS/USDA.
78. U.S. Department of Agriculture. 2003. Interpretive Summary: Quan-

- titative assessment of the relative risk to public health from food-borne *L. monocytogenes* among selected categories of ready-to-eat foods. CFSAN/FDA, FSIS/USDA.
79. USDA, ARS, NAA, ERRC, MFSRU Pathogen Modeling Program. PMP 7.0 Version number: 1.1.1433.15425. Platform: MSWindows 98, NT4 and newer. Available at: <http://www.arserrc.gov/mfs/pathogen.htm>
 80. Vangtal, A., and E. Hammond. 1986. Relation of the flavors of Swiss cheese to chemical parameters. *J. Dairy Sci.* 69 (suppl. 1):81 (abstract).
 81. Villani, F., O. Pepe, G. Mauriello, G. Moschetti, L. Sannino, and S. Coppola. 1996. Behavior of *L. monocytogenes* during the traditional manufacture of water-buffalo Mozzarella cheese. *Lett. Appl. Microbiol.* 22:357–360.
 82. White, C. H., and E. W. Custer. 1976. Survival of *Salmonella* in Cheddar cheese. *J. Milk Food Technol* 39(5): 328–331.
 83. Wijtzes, T., P. J. McClure, M. H. Zwietering, and T. A. Roberts. 1993. Modeling bacterial growth of *L. monocytogenes* as a function of water activity, pH and temperature. *Intern. J. Food Micro* 18:139–149.
 84. Wood, D. S., D. L. Collins-Thompson, D. M. Irvine, and A. N. Myhr. 1984. Source and persistence of *Salmonella muenster* in naturally contaminated Cheddar cheese. *J. Food Prot.* 47(1):2–22.
 85. Yousef, A. E., and E. H. Marth. 1988. Behavior of *L. monocytogenes* during the manufacture and storage of Colby cheese. *J. Food Prot.* 51(1): 12–15.
 86. Yousef, A. E., and E. H. Marth. 1990. Fate of *L. monocytogenes* during the manufacture and ripening of Parmesan cheese. *J. Dairy Sci.* 73(12):3351–3356.
 87. Zhao, T., M. P. Doyle, and R. E. Besser. 1993. Fate of enterohemorrhagic *Escherichia coli* O157:H7 in apple cider with and without preservatives. *Appl. Environ. Microbiol.* 59:2526–2530.